四川大学学报（医学版） 2026，57（2）：419－426
J Sichuan Univ（Med Sci） doi: 10.12182/20260360110

在线全文

创伤性凝血病早期风险预测模型的构建*

徐 旭[1,2]，贺美娜[1]，邵 敏[1]，刘 欣[1,2]，易 起[3]，张炳炎[3]，黄 莹[3]，谭超超[1,2△]

1. 湖南省人民医院（湖南师范大学附属第一医院）检验科（长沙 410005）；2. 湖南师范大学医学部（长沙 410013）；
3. 湖南省人民医院（湖南师范大学附属第一医院）急诊科（长沙 410005）

【摘要】 **目的** 基于前瞻性收集的创伤患者入院早期临床资料和实验室数据，构建并验证用于早期评估急诊创伤患者并发创伤性凝血病（trauma-induced coagulopathy, TIC）的风险预测模型。**方法** 本研究分析了2024年1月–2024年12月期间收治的285例急诊创伤患者的临床资料和实验室数据。按7∶3比例将患者随机分为训练集（n=199）与测试集（n=86）。通过单因素与多因素logistic回归分析筛选TIC的独立预测因素并构建风险预测模型。采用受试者工作特征曲线下面积（area under the curve, AUC）评估模型的诊断效能，使用Bootstrap法绘制校准曲线以评估校准度，并利用决策曲线分析（decision curve analysis, DCA）评价其临床净获益。**结果** 多因素logistic回归分析确定头部创伤、平均动脉压（mean arterial pressure, MAP）、凝血酶原时间（prothrombin time, PT）和凝血酶时间（thrombin time, TT）为TIC的独立预测因素，并成功构建预测模型。该模型在训练集中的AUC为0.804〔95%置信区间（confidence interval, CI）：0.737～0.871〕，在测试集中的AUC为0.847（95%CI：0.767～0.927）。校准曲线显示模型预测概率与实际概率高度一致，DCA表明该模型在较宽的风险阈值范围内（0.2～1.0）具有显著的临床净获益。**结论** 本研究成功构建并验证了TIC风险预测模型，该模型对急诊创伤患者并发TIC具有良好的早期预测效能。

【关键词】 创伤与损伤 凝血障碍 Logistic模型 风险评估

Construction of an Early-stage Risk Prediction Model for Trauma-Induced Coagulopathy

XU Xu[1,2], *HE Meina*[1], *SHAO Min*[1], *LIU Xin*[1,2], *YI Qi*[3], *ZHANG Bingyan*[3], *HUANG Ying*[3], *TAN Chaochao*[1,2△]. 1. *Department of Clinical Laboratory, Hunan Provincial People's Hospital (The First Affiliated Hospital of Hunan Normal University), Changsha 410005, China*; 2. *Department of Medical, Hunan Normal University, Changsha 410013, China*; 3. *Department of Emergency Medicine, Hunan Provincial People's Hospital (The First Affiliated Hospital of Hunan Normal University), Changsha 410005, China*

△ Corresponding author, E-mail: w2236@hunnu.edu.cn

This study was supported by the University-Industry Cooperation Collaborative Education Program of the Ministry of Education (No. 231107010130740), the High-level Talents Project of Hunan Provincial Health Commission (No. R2023087), the Innovation Team Project of Hunan Provincial People's Hospital (No. KCT202402), the Project of Hunan Institute of Geriatrics (No. 20240307-1014), the Key R&D Program of Hunan Province (No. 2025JK2130), the Excellent Young Scholars Project of Hunan Provincial Department of Education (No. 24B0073), the Changsha Municipal Natural Science Foundation (No. kq2502185), the Project of Hunan Provincial Health Commission (No. 20254678), the China Postdoctoral Science Foundation (No. 2025M781976), and the Postgraduate Scientific Research Innovation Project of Hunan Provincial Department of Education (No. CX20250819).

[**Abstract**] **Objective** Based on prospectively collected early clinical and laboratory data from trauma patients at admission, a risk prediction model for the early assessment of trauma-induced coagulopathy (TIC) in emergency trauma patients was constructed and validated. **Methods** This study analyzed the clinical data and laboratory results of 285 emergency trauma patients admitted between January 2024 and December 2024. The patients were randomly divided into a training set (n = 199) and a test set (n = 86) at a 7∶3 ratio. Univariate and multivariate logistic regression analyses were performed to identify independent predictors of TIC and to construct a risk prediction model. The diagnostic efficacy of

* 教育部产学合作协同育人项目（No. 231107010130740）、湖南省卫生健康委员会高层次人才项目（No. R2023087）、湖南省人民医院创新团队项目（No. KCT202402）、湖南省老年医学研究所项目（No. 20240307-1014）、湖南省重点研发计划项目（No. 2025JK2130）、湖南省教育厅优秀青年项目（No. 24B0073）、长沙市自然科学基金项目（No. kq2502185）、湖南省卫生健康委员会项目（No. 20254678）、中国博士后科学基金项目（No. 2025M781976）和湖南省教育厅研究生科研创新项目（No. CX20250819）资助

△ 通信作者，E-mail: w2236@hunnu.edu.cn

出版日期：2026-03-20

the model was evaluated by the area under the receiver operating characteristic curve (AUC). The calibration curve was plotted using the Bootstrap method to assess calibration, and the clinical net benefit was evaluated by decision curve analysis (DCA). **Results** Multivariate logistic regression analysis identified head trauma, mean arterial pressure (MAP), prothrombin time (PT), and thrombin time (TT) as independent predictors of TIC, and a predictive model was developed. The AUC of the model was 0.804 (95% CI: 0.737-0.871) in the training set and 0.847 (95% CI: 0.767-0.927) in the test set. The calibration curve showed a high level of agreement between the predicted and actual probabilities. DCA indicated that the model provided significant clinical net benefit across a broad range of risk thresholds (0.2-1.0). **Conclusion** This study developed and validated a TIC risk prediction model that demonstrated excellent early predictive efficacy for TIC in emergency trauma patients.

[Key words] Wounds and injuries Blood coagulation disorders Logistic models Risk assessment

创伤是全球范围内导致死亡的主要原因之一[1]，早期死亡最常见的原因是创伤引起的无法控制的出血[2-3]，这与急诊创伤所诱发的创伤性凝血病(trauma-induced coagulopathy, TIC)密切相关[4]。TIC是严重创伤后早期发生的急性凝血功能紊乱综合征[5]，其病理机制复杂，常涉及内皮损伤、凝血因子耗竭、纤溶亢进及代谢紊乱等多重因素[6]。在疾病早期，患者多处于低凝血期，临床表现为大量出血；在疾病晚期则转变为高凝血期，临床表现为与静脉血栓和多器官功能衰竭相关的过度凝血状态[6-7]。TIC与患者死亡率升高、输血需求增加及多器官功能障碍的发生密切相关[4, 8]。流行病学研究表明，约25%～33%的重症创伤患者在入院时即存在TIC[9]，其病死率是无凝血障碍患者的4至6倍[10]。

因此，早期识别TIC对于及时干预、控制病情进展及改善预后至关重要[11]。然而，由于TIC的凝血功能紊乱特性，传统凝血功能检测对TIC的诊断价值存在一定的局限性，且无法在临床有限的时间内获得结果以指导治疗[12-13]。同时，目前临床缺乏统一的TIC诊断标准和实用的风险预测模型[7, 14]，导致临床实践中难以实现个体化风险评估与精准干预。为解决以上问题，本研究通过纳入一些新的指标，构建并验证了一种用于早期评估急诊创伤患者并发TIC风险的风险预测模型，以期为临床医生提供更及时、客观的辅助判断工具。

1 资料与方法

1.1 研究对象

本研究为一项前瞻性队列研究，纳入2024年1月–2024年12月湖南省人民医院收治的285例急诊创伤患者，收集患者入院早期临床资料及其血液样本。纳入标准：①严重创伤：损伤严重程度评分(injury severity score, ISS)≥16分或者简明损伤定级标准(abbreviated injury scale, AIS)最大值≥3；②创伤原因和部位不限；③受伤至入院时间<24 h；④有完整的临床资料。排除标准：①患有血液系统疾病，先天性或后天性凝血系统异常的患者；②有服用抗凝药物病史；③恶性肿瘤、严重肝硬化等原发疾病患者；④外院治疗后转至我院患者。TIC的诊断标准[15]：活化部分凝血活酶时间(activated partial thromboplastin time, APTT)>40 s、国际标准化比值(international normalized ratio, INR)>1.2、血小板(blood platelet, PLT)计数<100×10^9 L^{-1}、纤维蛋白原(fibrinogen, FIB)<2 g/L(符合其一)。评估创伤患者入院时的格拉斯哥昏迷量表(Glasgow Coma Scale, GCS)、创伤指数(trauma index, TI)、ISS和AIS。评分采用单盲的形式，由两名及以上医生进行评分，结果取平均值。285例患者按照7∶3比例分为训练集(n=199)与测试集(n=86)。本研究根据赫尔辛基宣言进行，并由湖南省人民医院医学伦理委员会批准(批准号[2024]-01)。

1.2 临床资料收集

采集患者入院后(创伤发生<24 h)临床资料，包括：①基本资料：年龄与性别；②创伤特征：创伤原因(交通事故、高处坠落、锐器刺伤、暴力袭击及其他)与创伤部位(头颈部、胸部、腹部/骨盆、四肢)；③入院生命体征：心率、血氧饱和度与平均动脉压(mean arterial pressure, MAP)；④创伤评分：TI、GCS、ISS及AIS最大值；⑤基础疾病：糖尿病与高血压病史。

1.3 实验室检测

患者入院后(创伤发生<24 h)采集适量肘静脉血于EDTA抗凝管和枸橼酸钠抗凝管(1∶9)中，采集桡动脉或股动脉血于预设肝素锂的专用动脉血气针中。血液样本实验室检测内容包括：①凝血功能：凝血酶原时间(prothrombin time, PT)、PT-INR、凝血酶原活动度(PT%)、APTT、凝血酶时间(thrombin time, TT)和FIB采用凝固法检测，D-二聚体(D-dimer, DD)和纤维蛋白(原)降解产物(fibrin/fibrinogen degradation products, FDP)采用免疫比浊法检测，抗凝血酶-Ⅲ(antithrombin-Ⅲ, AT-Ⅲ)采用发色底物法检测(MDC7500全自动凝血分析仪，

北京九强)；②血常规: 白细胞、中性粒细胞、淋巴细胞、PLT计数及血红蛋白采用流式细胞术/电阻抗法检测(XN-10[B4]全自动血液分析仪，日本希森美康株式会社)；③动脉血气分析: pH值、碱剩余(base excess, BE)及乳酸水平采用电极法检测(PAPIDPOINT 500血气分析仪，美国西门子)；④C反应蛋白(C-reactive protein, CRP)采用免疫比浊法检测(PA-990特定蛋白分析仪，深圳普门科技)。

1.4　统计学方法

使用SPSS 26对临床资料进行分析。缺失值采用多重插补法进行5次插补。符合正态分布的定量资料以$\bar{x} \pm s$表示，组间比较采用独立样本t检验。不符合正态分布的定量资料则以中位数(P_{25}, P_{75})表示，组间比较采用非参数检验。定性资料表示为例数(百分数)，组间比较采用卡方检验。$P<0.05$ 为差异有统计学意义。

鉴于结局变量为二分类变量，故采用logistic回归分析。将具有统计学意义的指标纳入后续的多因素逐步logistic回归(向前: LR)构建风险预测模型。

使用R 4.4.3软件进行模型的决策曲线分析(decision curve analysis, DCA)，评估模型的临床价值。绘制模型列线图，并使用Bootstrap方法重复采样1000次进行内部验证。

使用Graphpad Prism10绘制模型受试者工作特征(receiver operating characteristic, ROC)曲线，评估模型的诊断能力，通过Omnibus检验和Hosmer-Lemeshow检验评估模型是否成立及其拟合效果。

2　结果

2.1　训练集与测试集临床资料比较

对训练集与测试集患者的临床资料进行了全面比较，具体见网络资源附件附表1。统计分析表明，两组患者在基线资料、创伤评分及实验室指标等所有临床资料上，差异均无统计学意义。这表明，训练集与测试集患者具有高度的可比性，确保了后续模型构建与验证过程的可靠性。

2.2　训练集中TIC组与非TIC组临床资料比较

与非TIC组相比，TIC组患者入院时表现出更低的MAP和更严重的创伤评分($P<0.05$)。在创伤部位上，TIC组患者中头颈部创伤与胸部创伤的发生比例高于非TIC组($P<0.05$)，表明这些部位的严重创伤可能与TIC的发生存在关联。在实验室指标中，两组间的差异尤为明显: TIC组患者的PT、PT-INR、APTT、TT高于非TIC组患者，PT%、FIB和PLT计数则降低，差异均有统计学意义($P<0.05$)。此外，根据动脉血气分析结果，TIC组患者呈现出更严重的酸中毒状态，具体表现为pH值更低、BE负值更大以及乳酸水平更高，差异均有统计学意义($P<0.001$)。见表1。

2.3　影响急诊创伤患者并发TIC的多因素logistic回归

基于单因素分析结果，筛选出TIC组与非TIC组间差异有统计学意义的变量，并将这些变量纳入多因素logistic回归分析，得到了包括头部创伤、MAP、PT、TT这4个关键变量在内的急诊创伤患者并发TIC的风险预测模型。

虽然逐步回归显示MAP并无统计学意义，但基于模型的实际应用价值考虑，保留了该指标。同时本研究通过比较纳入与排除该变量后的模型性能发现，纳入变量MAP后，虽然模型的曲线下面积(area under the curve, AUC)值仅从0.799提升至0.804，但显著改善了模型的校准度，Hosmer-Lemeshow检验从$P<0.05$(模型拟合不佳)转为$P>0.05$(模型拟合良好)。最终本研究认为将MAP纳入模型是合理的。

多因素分析结果显示，MAP为TIC发生的独立保护因素，头部创伤、PT、TT均为独立危险因素，见表2。

2.4　急诊创伤患者并发TIC的风险预测模型效能评估与验证

在训练集中，得到的急诊创伤患者并发TIC的风险预测模型AUC值为0.804〔95%置信区间(confidence interval, CI): 0.737 ~ 0.871〕，诊断效能良好(图1)。Omnibus检验结果显示模型成立($P<0.05$)；同时，Hosmer-Lemeshow检验结果表明该模型拟合效果良好($P>0.05$)。在测试集中对新模型进行验证，结果显示新模型的AUC值达到0.847(95%CI: 0.767 ~ 0.927)，说明新模型在测试集中同样具有良好的诊断效能(图1)。

基于急诊创伤患者TIC风险预测模型绘制列线图，将模型可视化(图2)。列线图整合了模型中的所有预测因素，方便临床上及时根据患者临床指标快速评估其发生TIC的风险，相关示例结果见网络资源附件附表2。同时为了验证模型的校准能力，本研究采用Bootstrap法重复抽样1000次，绘制校准曲线，校准曲线显示模型的原始曲线趋近于校准曲线(模型预测概率与实际概率较为接近)，表明模型具有良好的校准度(图3)。

此外，为评估该预测模型在临床实践中的适用性与效益，本研究进一步进行了DCA(图4)。结果显示风险阈值在0.2 ~ 1.0范围内，该模型的临床净获益(Net Benefit)始终高于“全部干预”(ALL)和“全不干预”(None)两条参考策略线。结果表明，基于该模型进行临床决策可在较宽的风险判断范围内提供更高的临床净获益，具备良好的临床价值。本研究建立的TIC风险预测模型具有良好

表 1 训练集中TIC组与非TIC组间的临床数据比较

Table 1 Comparison of clinical data between the TIC group and the non-TIC group within the training set

Clinic data	TIC (n = 72)	Non-TIC (n = 127)	$t/\chi^2/z$	P
Sex/case (%)			0.032	0.858
Male	53 (73.6)	92 (72.4)		
Female	19 (26.4)	35 (27.6)		
Age/yr., M (P_{25}, P_{75})	51.5 (39.3, 60.0)	54.0 (41.0, 64.0)	−0.983	0.326
Diabetes/case (%)	9 (12.5)	9 (7.1)	1.637	0.201
Hypertension/case (%)	10 (13.9)	23 (18.1)	0.592	0.442
Heart rate/min^{-1}, M (P_{25}, P_{75})	84.5 (72.3, 97.2)	85.0 (75.0, 95.0)	−0.195	0.846
Oxygen saturation/%, M (P_{25}, P_{75})	99.0 (96.0, 99.0)	98.0 (97.0, 99.0)	−0.121	0.904
Mean arterial pressure/mmHg, M (P_{25}, P_{75})	92.7 (83.4, 101.6)	96.9 (88.0, 109.0)	−2.409	0.016
Trauma score (M [P_{25}, P_{75}])				
TI	10.0 (7.3, 13.8)	9.0 (6.0, 11.0)	−2.732	0.006
GCS	14.5 (10.0, 15.0)	15.0 (15.0, 15.0)	−4.038	< 0.001
ISS	16.5 (10.8, 24.0)	13.0 (9.0, 17.0)	−3.762	< 0.001
AIS	3.0 (3.0, 4.0)	3.0 (3.0, 3.0)	−3.894	< 0.001
Causes of trauma/case (%)				
Traffic accident	42 (58.3)	66 (52.0)	0.750	0.386
Falling from a height	27 (37.5)	46 (36.2)	0.032	0.857
Sharp instrument stab wound	1 (1.4)	5 (3.9)	0.335	0.563
Violent attack	0 (0.0)	1 (0.8)		1.000
Other	2 (2.8)	7 (5.5)	0.288	0.591
Trauma site/case (%)				
Head and neck	50 (69.4)	50 (39.4)	16.624	< 0.001
Chest	42 (58.3)	52 (40.9)	5.574	0.018
Abdomen/Pelvis	19 (26.4)	28 (22.0)	0.480	0.488
Limbs	27 (37.5)	50 (39.4)	0.068	0.795
PT/s, M (P_{25}, P_{75})	12.9 (11.7, 14.5)	11.9 (10.8, 12.7)	−4.717	< 0.001
PT-INR (M [P_{25}, P_{75}])	1.1 (1.0, 1.2)	1.0 (0.9, 1.1)	−5.319	< 0.001
PT% (M [P_{25}, P_{75}])	85.6 (73.6, 100.4)	97.8 (86.7, 109.3)	−4.479	< 0.001
APTT/s, M (P_{25}, P_{75})	27.0 (23.2, 31.4)	24.5 (22.7, 26.3)	−3.556	< 0.001
TT/s, M (P_{25}, P_{75})	15.5 (13.2, 18.5)	14.7 (12.4, 16.9)	−2.148	0.032
FIB/(g/L), M (P_{25}, P_{75})	1.7 (1.6, 1.9)	2.5 (2.2, 3.0)	−10.453	< 0.001
DD/(mg/L), M (P_{25}, P_{75})	3 186.0 (197.8, 8 863.8)	2 062.0 (140.0, 7 200.7)	−1.409	0.159
FDP/(mg/L), M (P_{25}, P_{75})	71.8 (26.3, 114.3)	80.5 (32.7, 128.8)	−0.635	0.525
AT-3/%, M (P_{25}, P_{75})	98.5 (89.8, 103.6)	99.6 (92.9, 104.0)	−1.335	0.182
White blood cells/(× 10^9 L^{-1}), M (P_{25}, P_{75})	12.0 (8.5, 16.3)	10.8 (8.9, 14.2)	−1.123	0.261
Neutrophils/(× 10^9 L^{-1}), M (P_{25}, P_{75})	8.0 (5.6, 13.0)	8.0 (5.6, 12.1)	−0.517	0.605
Lymphocyte/(× 10^9 L^{-1}), M (P_{25}, P_{75})	2.3 (1.4, 3.5)	1.9 (1.1, 3.0)	−1.237	0.216
Platelet/(× 10^9 L^{-1}), M (P_{25}, P_{75})	196.5 (156.3, 227.3)	230.0 (184.0, 273.0)	−3.763	< 0.001
Hemoglobin/(g/L), M (P_{25}, P_{75})	135.0 (119.5, 147.0)	138.0 (126.0, 150.0)	−1.457	0.145
CRP/(mg/L), M (P_{25}, P_{75})	5.1 (0.5, 16.9)	7.1 (0.9, 17.6)	−0.660	0.509
pH (M [P_{25}, P_{75}])	7.3 (7.3, 7.4)	7.4 (7.3, 7.4)	−3.607	< 0.001
BE/(mmol/L), M (P_{25}, P_{75})	−4.4 (−6.9, −2.6)	−2.9 (−5.0, −0.5)	−3.772	< 0.001
Lactic acid/(mmol/L), M (P_{25}, P_{75})	5.4 (3.9, 6.9)	4.1 (2.2, 5.9)	−3.488	< 0.001

M: median; TI: trauma index; GCS: Glasgow Coma Scale; ISS: injury severity score; AIS: abbreviated injury scale; PT: prothrombin time; PT-INR: prothrombin time-international normalized ratio; PT%: prothrombin time activity (percentage); APTT: activated partial thromboplastin time; TT: thrombin time; FIB: fibrinogen; DD: D-dimer; FDP: fibrin degradation products; AT-3: antithrombin Ⅲ; CRP: C-reactive protein; BE: base excess. 1 mmHg = 0.133 kPa.

表 2　TIC组与非TIC组间的多变量logistic回归分析

Table 2　Multivariate logistic regression analysis was conducted between the TIC group and the non-TIC group

Predictive factor	β	SE	Wald	P	OR	95% CI
HNT	1.335	0.369	13.062	< 0.001	3.798	1.842-7.833
MAP*	−0.021	0.012	3.159	0.076	0.979	0.956-1.002
PT	0.726	0.139	27.105	< 0.001	2.066	1.572-2.715
TT	0.254	0.071	12.669	< 0.001	1.289	1.121-1.482

HNT: head and neck trauma; MAP: mean arterial pressure; PT: prothrombin time; TT: thrombin time; SE: standard error; OR: odds ratio. * Stepwise regression showed that MAP was not statistically significant, but this indicator was retained based on the practical application value of the model. After including the variable MAP, the AUC value of the model increased from 0.799 to 0.804, and the calibration of the model improved. The Hosmer-Lemeshow test changed from $P < 0.05$ (poor model fit) to $P > 0.05$ (good model fit).

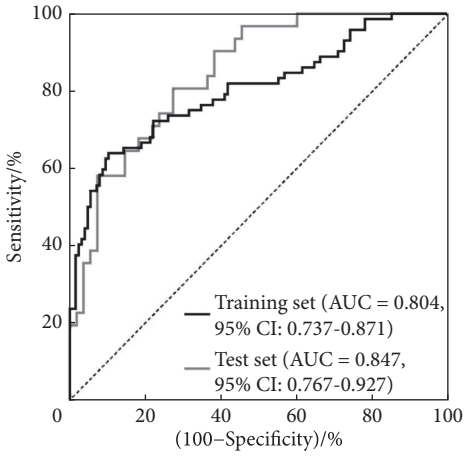

图 1　训练集和测试集的TIC风险预测模型的ROC曲线

Fig 1　The ROC curves of the TIC risk prediction models for the training set and the test set

AUC: area under the curve. Training set: n = 199; test set: n = 86.

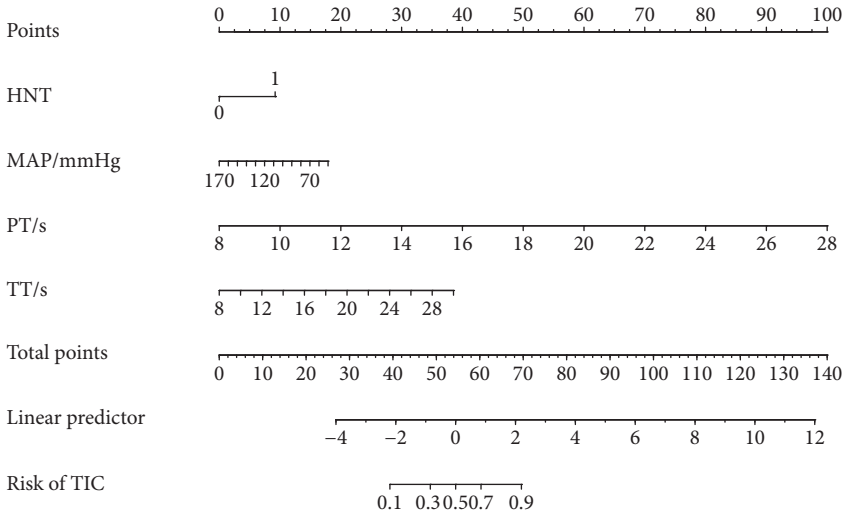

图 2　TIC风险预测模型的列线图

Fig 2　Nomogram of the TIC risk prediction model

HNT: head and neck trauma; MAP: mean arterial pressure; PT: prothrombin time; TT: thrombin time; TIC: trauma-induced coagulopathy. 1 mmHg = 0.133 kPa.

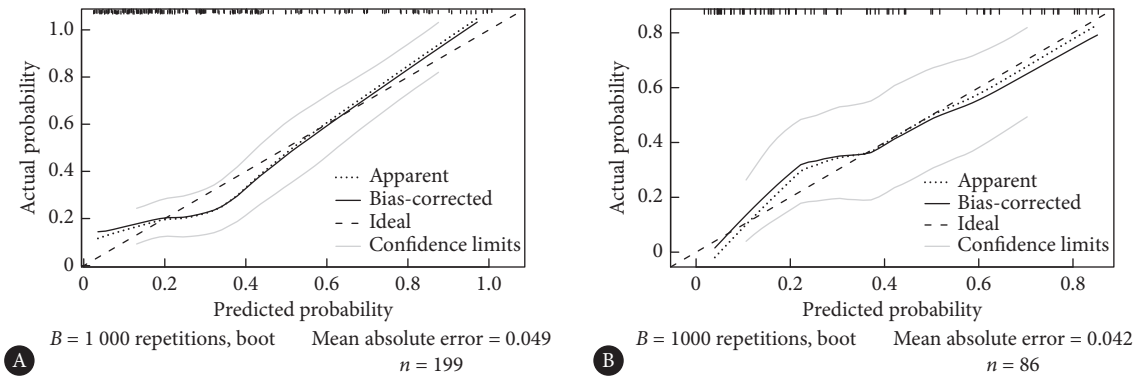

图 3　TIC风险预测模型的校准曲线

Fig 3　Calibration curve of the TIC risk prediction model

Calibration curves of the TIC risk prediction model for the training set (A) and for the test set (B). The calibration curve (dashed line) represents the performance of the model on the original data, while the calibration curve after 1 000 bootstrap resamplings (solid line) represents the corrected performance of the model.

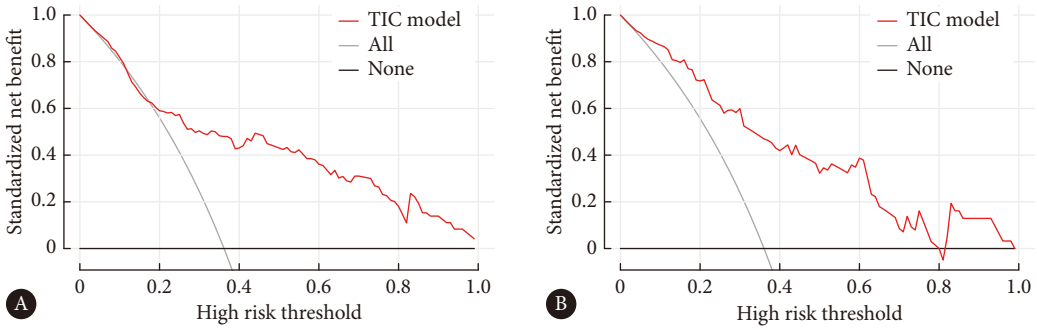

图 4 TIC风险预测模型的决策曲线分析

Fig 4 Decision curve analysis of the TIC risk prediction model

Decision curve analysis (DCA) of the trauma-induced coagulopathy (TIC) risk prediction model for the training set (A) and for the test set (B). The *X*-axis represents the high-risk threshold, and the *Y*-axis represents the standardized net benefit. The horizontal black line (None) represents the assumption that all patients do not receive treatment, while the gray line (All) represents the assumption that all patients receive treatment. The red line indicates the clinical net benefit of the model.

的早期预测效能，当预测风险≥20%时建议启动临床干预，有助于个体化治疗决策。

3 讨论

本研究通过分析前瞻性纳入的急诊创伤患者临床和实验室数据，成功构建并验证了一个基于创伤后24 h内指标的TIC风险预测模型。需注意的是，模型中纳入的指标MAP在逐步回归分析中其*P*值> 0.05。出现这一现象可能的原因是MAP与模型中其他变量（如PT、TT）可能存在潜在的生物学关联，当这些凝血指标进入模型后，它们解释了TIC发病中的较大部分，导致MAP的独特贡献被稀释，其回归系数虽具有临床意义的效应量，但未跨越显著性阈值；其次可能是受样本量限制，不足以检测出MAP在调整其他变量后的微弱独立效应。经综合考虑，并结合Hosmer-Lemeshow检验结果，模型最终纳入了头部创伤、MAP、PT和TT四个预测因素，该模型在训练集和测试集中均显示出良好的诊断效能（AUC> 0.8）和校准度。此外，模型在风险阈值0.2 ~ 1.0范围内提供了更高的临床净获益，具备良好的临床价值，可以早期评估急诊创伤患者的TIC风险。结合临床实际，本研究建议将20%（0.2）作为启动TIC针对性干预的参考阈值：当模型预测的TIC风险≥20%时，应考虑早期实施干预措施（如启动大量输血方案、输注氨甲环酸、补充凝血因子等）；当风险< 20%时，则暂不进行特殊干预，但仍需密切监测。

相较于其他部位创伤，头部创伤患者并发TIC的风险更高，是TIC的独立危险性因素。在发生创伤性脑损伤后，大脑组织中表达的组织因子大量释放入血[16]，通过外源性凝血途径启动全身凝血激活，最终导致血小板耗竭、凝血因子消耗和纤溶亢进[17-18]。有研究发现，在严重创伤性脑损伤患者中，随着头部创伤严重程度的增加，凝血病的发生率逐步增加[19-20]。同时在没有失血性休克或多系统损伤的情况，仅创伤性脑损伤就足以诱发严重的血小板功能障碍及TIC[20-21]。本研究将头部创伤纳入TIC风险预测模型中，具有坚实的病理生理学基础。

MAP的高低也与TIC的发生密切相关[8]，是TIC的独立保护性因素。低MAP患者体内有效循环血量减少，组织灌注减少，继而引发代谢性酸中毒和乳酸堆积[22]。在本研究中，伴发TIC的急诊创伤患者的pH值、BE和乳酸含量都证实了这一点。酸中毒被公认为是TIC的关键致病因素，会加剧内皮细胞、免疫系统、血小板和凝血系统的激活[6]。有临床研究报道，低MAP和高休克指数（SI）是TIC发生的危险因素，是创伤患者并发TIC的重要预测指标[23]。同时有研究人员发现，SI还能作为创伤患者死亡的预测指标，是急诊创伤患者预后不良的独立危险性因素[24]。低MAP作为休克和组织灌注不足的替代指标，其纳入预测模型强调了早期复苏和纠正低灌注对于预防TIC的重要性[25]。

本预测模型还纳入了常规凝血功能检测中的两个关键指标——PT和TT，它们从不同层面揭示了TIC的病理生理状态，是早期识别凝血功能障碍的灵敏指标。PT是反映外源性凝血途径功能的核心指标，PT的延长主要提示凝血因子Ⅱ、Ⅴ、Ⅶ、Ⅹ的合成不足或大量消耗[26]。在创伤早期，患者大量出血和发生弥漫性血管内凝血导致凝血因子被大量消耗[27]。而TT是反映人体共同凝血途径最后阶段的核心指标。TT的延长则主要提示FIB水平低下或功能异常，或者FDP的增多。在本研究中，伴发TIC的急诊创伤患者的FIB明显更少。在创伤早期，患者的大出血和弥漫性血管内凝血也会导致FIB的大量消耗[27]。PT和TT的显著延长共同提示创伤患者早期止血能力的崩溃。创伤患者无法有效形成血凝块，难以控制出血，表

现为TIC早期的低凝血期(出血期),严重危及患者生命[6]。有研究表明,PT还可以作为创伤患者不良预后的独立预测因子[28]。本研究结果证实了这一点,TIC患者的PT和TT均显著延长,符合TIC早期的低凝血期表现。也证明及时补充新鲜冰冻血浆以纠正凝血因子缺乏,或输注冷沉淀/纤维蛋白原浓缩物以提升FIB水平对于TIC患者是必要的[29]。

本研究存在一定的局限性,仅为单中心研究,可能存在选择偏倚和信息偏倚。未来需要多中心研究进行外部验证,同时扩大样本量来增强模型的普适性和临床适用价值。

综上所述,本研究基于急诊创伤患者创伤后24 h内的临床资料和实验室检测结果,纳入了头部创伤、MAP、PT和TT这四个在临床上易获取的指标,构建了一个性能良好的TIC风险预测模型。为临床早期识别TIC患者提供了基础,能有效改善患者预后。

*　　*　　*

作者贡献声明　徐旭负责论文构思、数据审编、正式分析、经费获取、调查研究、研究方法、验证、可视化、初稿写作和审读与编辑写作,贺美娜负责调查研究和提供资源,邵敏负责经费获取,刘欣负责调查研究,易起和张炳炎负责提供资源,黄莹负责研究项目管理和提供资源,谭超超负责论文构思、经费获取、调查研究、研究方法、研究项目管理、提供资源、监督指导和审读与编辑写作。所有作者已经同意将文章提交给本刊,且对将要发表的版本进行最终定稿,并同意对工作的所有方面负责。

Author Contribution　XU Xu is responsible for conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, validation, visualization, writing--original draft, and writing--review and editing. HE Meina is responsible for investigation and resources. SHAO Min is responsible for funding acquisition. LIU Xin is responsible for investigation. YI Qi and ZHANG Bingyan are responsible for resources. HUANG Ying is responsible for project administration and resources. TAN Chaochao is responsible for conceptualization, funding acquisition, investigation, methodology, project administration, resources, supervision, and writing--review and editing. All authors consented to the submission of the article to the Journal. All authors approved the final version to be published and agreed to take responsibility for all aspects of the work.

利益冲突　所有作者均声明不存在利益冲突

Declaration of Conflicting Interests　All authors declare no competing interests.

参　考　文　献

[1] MICLAU T, BALOGH Z J, MICLAU K R, *et al*. Trauma systems: a global comparison. OTA Int, 2025, 8(3 Suppl): e376. doi: 10.1097/OI9.0000000000000376.

[2] KALKWARF K J, DRAKE S A, YANG Y, *et al*. Bleeding to death in a big city: An analysis of all trauma deaths from hemorrhage in a metropolitan area during 1 year. J Trauma Acute Care Surg, 2020, 89(4): 716-722. doi: 10.1097/TA.0000000000002833.

[3] MOORE H B, MOORE E E, CHAPMAN M P, *et al*. Plasma-first resuscitation to treat haemorrhagic shock during emergency ground transportation in an urban area: a randomised trial. Lancet, 2018, 392(10144): 283-291. doi: 10.1016/S0140-6736(18)31553-8.

[4] BROHI K, SINGH J, HERON M, *et al*. Acute traumatic coagulopathy. J Trauma, 2003, 54(6): 1127-1130. doi: 10.1097/01.TA.0000069184.82147.06.

[5] CAPPONI A, ROSTAGNO C. Trauma-Induced Coagulopathy: A Review of Specific Molecular Mechanisms. Diagnostics (Basel), 2025, 15(11): 1435. doi: 10.3390/diagnostics15111435.

[6] MOORE E E, MOORE H B, KORNBLITH L Z, *et al*. Trauma-induced coagulopathy. Nat Rev Dis Primers, 2021, 7(1): 30. doi: 10.1038/s41572-021-00264-3.

[7] MOORE H B, GANDO S, IBA T, *et al*. Defining trauma-induced coagulopathy with respect to future implications for patient management: Communication from the SSC of the ISTH. J Thromb Haemost, 2020, 18(3): 740-747. doi: 10.1111/jth.14690.

[8] REED C R, CURRY N, JUFFERMANS N P, *et al*. Hemostatic abnormalities after trauma resuscitation: challenges and strategies in caring for the critically injured patient. Ann Intensive Care, 2025, 15(1): 163. doi: 10.1186/s13613-025-01587-0.

[9] KORNBLITH L Z, MOORE H B, COHEN M J. Trauma-induced coagulopathy: the past, present, and future. J Thromb Haemost, 2019, 17(6): 852-862. doi: 10.1111/jth.14450.

[10] BROHI K, COHEN M J, GANTER M T, *et al*. Acute coagulopathy of trauma: hypoperfusion induces systemic anticoagulation and hyperfibrinolysis. J Trauma, 2008, 64(5): 1211-1217; discussion 1217. doi: 10.1097/TA.0b013e318169cd3c.

[11] 中国医师协会急诊医师分会, 解放军急救医学专业委员会, 北京急诊医学学会, 等. 院前创伤急救止血专家共识(2025年版). 中华急诊医学杂志, 2025, 34(4): 469-477. doi: 10.3760/cma.j.issn.1671-0282.2025.04.003.
Emergency Physicians Branch of Chinese Medical Doctor Association, Professional Committee of Emergency Medicine of the Chinese People's Liberation Army, Beijing Society of Emergency Medicine, *et al*. Expert consensus on pre-hospital trauma emergency hemostasis (2025 edition). Chinese Journal of Emergency Medicine, 2025, 34(4): 469-477. doi: 10.3760/cma.j.issn.1671-0282.2025.04.003.

[12] PANDYA A P, GONZALEZ I J, KHAN M S, *et al*. Optimizing hemorrhage management in trauma: integrative roles of tranexamic acid and tissue plasminogen activator-augmented viscoelastic testing. J Thromb Thrombolysis, 2025. doi:10.1007/s11239-025-03194-z.

[13] HUTSPARDOL S, BORJA T, KROEKER J, *et al*. Comparison of conventional coagulation tests and ROTEM in identifying trauma-induced coagulopathy for massive haemorrhage protocol activation. Transfus Med, 2025, 35(3): 266-274. doi: 10.1111/tme.13128.

[14] PERKINS Z B, YET B, MARSDEN M, *et al*. Early identification of trauma-induced coagulopathy: development and validation of a multivariable risk prediction model. Ann Surg, 2021, 274(6): e1119-e1128. doi: 10.1097/SLA.0000000000003771.

[15] 袁强, 虞剑, 陈建, 等. 颅脑创伤急性期凝血功能障碍诊治专家共识(2024版). 中华创伤杂志, 2024, 40(4): 310-322. doi: 10.3760/cma.j.cn501098-20231107-00293.
YUAN Q, YU J, CHEN J, *et al*. Expert consensus on diagnosis and treatment of coagulopathy in acute phase of craniocerebral trauma (2024 edition). Chinese Journal of Traumatology, 2024, 40(4): 310-322. doi: 10.3760/cma.j.cn501098-20231107-00293.

[16] FLETCHER-SANDERSJÖÖ A, SEBGHATI J, THELIN E P. Hemostatic disturbances in traumatic brain injury: from mechanism to management. Acta Neurochir (Wien), 2025, 167(1): 146. doi: 10.1007/s00701-025-06549-w.

[17] HOU H, QU Z, LIU R, *et al*. Traumatic brain injury: advances in coagulopathy (review). Biomed Rep, 2024, 21(5): 156. doi: 10.3892/br.2024.1844.

[18] LIU J, TIAN H L. Relationship between trauma-induced coagulopathy and progressive hemorrhagic injury in patients with traumatic brain injury. Chin J Traumatol, 2016, 19(3): 172-175. doi: 10.1016/j.cjtee.2016.01.011.

[19] LIESCHKE F, MARGGRANDER D T, KOHLHASE K D, *et al*.

Coagulopathy, injury severity and bleeding progression but not prior antiplatelet and anticoagulation therapies drive prognosis in patients with moderate to severe traumatic brain injury. Front Neurol, 2025, 16: 1592583. doi: 10.3389/fneur.2025.1592583.

[20] CASTELLINO F J, CHAPMAN M P, DONAHUE D L, *et al*. Traumatic brain injury causes platelet adenosine diphosphate and arachidonic acid receptor inhibition independent of hemorrhagic shock in humans and rats. J Trauma Acute Care Surg, 2014, 76(5): 1169-1176. doi: 10.1097/TA.0000000000000216.

[21] WAFAISADE A, LEFERING R, TJARDES T, *et al*. Acute coagulopathy in isolated blunt traumatic brain injury. Neurocrit Care, 2010, 12(2): 211-219. doi: 10.1007/s12028-009-9281-1.

[22] 中华医学会重症医学分会. 低血容量休克复苏指南(2007). 中国危重病急救医学, 2008, 20(3): 129-134. doi: 10.3321/j.issn:1003-0603.2008.03.002.
Chinese Medical Association Critical Care Medicine Branch. Guidelines for resuscitation of hypovolemic shock (2007). Chinese Critical Care Medicine, 2008, 20(3): 129-134. doi: 10.3321/j.issn:1003-0603.2008.03.002.

[23] 姚立军, 陈立娟, 耿欣, 等. 休克指数和纤维蛋白原对老年严重创伤患者并发创伤性凝血病的发生及预后价值. 中国老年学杂志, 2023, 43(7): 1619-1623. doi: 10.3969/j.issn.1005-9202.2023.07.023.
YAO L J, CHEN L J, GENG X, *et al*. Value of shock index and fibrinogen in the occurrence and prognosis of traumatic coagulopathy in elderly patients with severe trauma. Chinese Journal of Gerontology, 2023, 43(7): 1619-1623. doi: 10.3969/j.issn.1005-9202.2023.07.023.

[24] VANG M, ØSTBERG M, STEINMETZ J, *et al*. Shock index as a predictor for mortality in trauma patients: a systematic review and meta-analysis. Eur J Trauma Emerg Surg, 2022, 48(4): 2559-2566. doi: 10.1007/s00068-022-01932-z.

[25] ROSSAINT R, AFSHARI A, BOUILLON B, *et al*. The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition. Crit Care, 2023, 27(1): 80. doi: 10.1186/s13054-023-04327-7.

[26] ZAIDI S R H, ROUT P. Interpretation of blood clotting studies and values (PT, PTT, aPTT, INR, anti-Factor Xa, D-Dimer). StatPearls. Treasure Island (FL): StatPearls Publishing, 2025.

[27] 中华医学会血液学分会血栓与止血学组. 弥散性血管内凝血诊断中国专家共识(2017年版). 中华血液学杂志, 2017, 38(5): 361-363. doi: 10.3760/cma.j.issn.0253-2727.2017.05.001.
Thrombosis and Hemostasis Group, Hematology Branch of Chinese Medical Association. Chinese expert consensus on the diagnosis of disseminated intravascular coagulation (2017 version). Chinese Journal of Hematology, 2017, 38(5): 361-363. doi: 10.3760/cma.j.issn.0253-2727.2017.05.001.

[28] VETTER P, NIGGLI C, HAMBRECHT J, *et al*. Prothrombin time predicting time-dependent and risk-stratified mortality in polytrauma patients. Int J Emerg Med, 2025, 18(1): 69. doi: 10.1186/s12245-025-00841-3.

[29] 王蔚, 周愚, 童珊珊, 等. 创伤性凝血病: 低纤维蛋白原血症. 中国呼吸与危重监护杂志, 2019, 18(1): 103-107. doi: 10.7507/1671-6205.201802006.
WANG W, ZHOU Y, TONG S S, *et al*. Traumatic coagulopathy: hypofibrinogenemia. Chinese Journal of Respiratory and Critical Care Medicine, 2019, 18(1): 103-107. doi: 10.7507/1671-6205.201802006.

(2025 – 10 – 12收稿, 2026 – 02 – 21修回)

编辑 余 琳

开放获取 本文使用遵循知识共享署名—非商业性使用4.0国际许可协议(CC BY-NC 4.0), 详细信息请访问https://creativecommons.org/licenses/by-nc/4.0/。

OPEN ACCESS This article is licensed for use under Creative Commons Attribution-NonCommercial 4.0 International license (CC BY-NC 4.0). For more information, visit https://creativecommons.org/licenses/by-nc/4.0/.

© 2026《四川大学学报(医学版)》编辑部

Editorial Office of *Journal of Sichuan University (Medical Sciences)*